UNITED STATES DISTRICT COURT FOR THE NORTHERN DISTRICT OF OKLAHOMA

DOUG INGRAM, et al.,	)	
	)	
Plaintiffs,	)	
	)	
v.	)	**Case No. 04-CV-0287-CVE-PJC**
	)	
SOLKATRONIC CHEMICAL, INC., JEFF HANNIS, and AIR PRODUCTS AND CHEMICALS, INC., a Delaware corporation,	)	
	)	
Defendants.	)	

OPINION AND ORDER

This matter comes before the Court on the request of defendant Solkatronic Chemical, Inc. (hereinafter "defendant") to exclude the expert testimony offered by plaintiffs. The Court held a Daubert hearing at which the proposed experts for defendant and plaintiffs testified. The Court has before it all of the related documents (Dkt. ## 86-104, 109, 110). Also before the Court are the motions to strike the testimony of two of plaintiffs' experts on alternative, procedural grounds (Dkt. ## 52, 85).

I.

Plaintiffs'[1] claims arise from their alleged exposure to arsine gas, after a July 11, 2001 accidental release following the rupture of a cylinder at the Solkatronics facility located on the Port of Catoosa. Plaintiffs claim to have suffered a range of adverse health effects subsequent to the release, and attribute their symptoms to exposure to arsine gas on July 11. Approximately four years after the July 11 release, all of the plaintiffs complain of headaches, generalized fatigue and

[1] There is a total of 192 plaintiffs. The parties are proceeding to trial with an initial group of twelve plaintiffs: Biddle, Cardenas, Castro, Guerra, Haggard, Hinton, Ingram, Miller, Patton, Schnitzer, Shavers, and Sumter.

weariness, memory loss and other neurological dysfunction, and ongoing emotional distress. Plaintiffs have produced three experts--Dr. Richard Hastings, Dr. Robert Harrison, and Dr. Shayne Gad--to validate their claims of arsine-induced injury. Defendant challenges each of plaintiffs' experts under Rule 702 and have retained their own experts--Dr. William Banner, Dr. Dean Carter, and Dr. Steven Pike--for the purpose of rebutting the opinions of plaintiffs' experts[2].

II.

Rule 702 of the Federal Rules of Evidence governs the admission of expert testimony:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training or education may testify thereto in the form of opinion or otherwise if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

Fed. R. Evid. 702. Rule 702 both empowers and requires the trial court to act as a gatekeeper for proposed expert testimony. See Ralston v. Smith & Nephew Richards, Inc., 275 F.3d 965, 969 (10th Cir. 2001). The rule directs the trial court to assess, first, whether a proposed expert is qualified by "knowledge, skill, experience, training, or education" to render an opinion. Id. If the court finds the expert qualified, it must then determine whether the opinions offered by the expert are reliable under the principles laid out in Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993).

The Daubert reliability inquiry demands that a proposed expert's testimony be anchored in the knowledge and experience of the discipline of the proffered witness.[3] Id. at 597. The Daubert

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[2] Defendant's experts are not the subjects of a challenge under Rule 702.

[3] The Daubert framework also requires a showing of relevance. The relevance of the proposed testimony in this case is not at issue.

Court outlined four factors appropriate for a district court's consideration in assessing the admissibility of expert testimony: (1) whether a theory has been or can be tested or falsified; (2) whether the theory or technique has been subject to peer review and publication; (3) whether there are known or potential rates of error with regard to specific techniques; and (4) whether the theory or approach has "general acceptance." Id. at 593-94. District courts are not confined to those factors, however, and may consider others when appropriate. See, e.g., Kumho Tire Co. Ltd., v. Carmichael, 526 U.S. 137, 150 (1999) ("Daubert makes clear that the factors it mentions do not constitute a definitive checklist or test.") (internal quotations marks omitted); Bitler v. A.O. Smith Corp., 400 F.3d 1227, 1233 (10th Cir. 2004) ("[T]his list is neither definitive nor exhaustive and . . . a trial judge has wide discretion both in deciding how to assess an expert's reliability and in making a determination of that reliability.").

In a toxic tort case, a plaintiff must offer reliable evidence of both general and specific causation, that is, whether a substance can cause a particular injury and whether a substance caused an individual's injury in a particular case. Norris v. Baxter Healthcare Corp., 397 F.3d 878, 881 (10th Cir. 2005).

III.

Arsine is a colorless, flammable gas. While neither party challenges arsine's toxicity, the parties dispute the extent to which the mechanism of arsine injury documented in the existing literature on the subject is exclusive of other possibilities for arsine toxicity. Extant research identifies a mechanism of injury centered on the red blood cell. Because of arsine's affinity for hemoglobin, an oxygen-carrying molecule contained within the red blood cell, it tends to bind preferentially to the red blood cell upon inhalation, and its chemical interaction with the red blood

cell produces a process called hemolysis, or the breaking apart of the red blood cell. Based upon one's view of the applicable science, hemolysis is either one of many possible results of arsine exposure or the lodestar marker of arsine toxicity.[4]

Hemolysis prompts a chain reaction that noticeably alters the makeup of an exposed individual's blood. Hemoglobin is released into the blood stream, decreasing the amount of hemoglobin bound within the red blood cells, causing a concomitant increase in the amount of hemoglobin moving freely within the blood stream, also known as plasma free hemoglobin. Also associated with hemolysis is a decrease in haptoglobin, an agent responsible for the binding of free hemoglobin in the bloodstream. As the released hemoglobin overwhelms the haptoglobin's ability to collect the products of the lysed red blood cells, haptoglobin levels drop. Finally, because hemolysis is, by definition, the destruction of the red blood cell, hemolysis may produce a reduced hematocrit, the proportion of the bloodstream made up of red blood cells. The literature on the subject of arsine toxicity documents a so-called "triad" of symptoms caused by hemolysis: abdominal pain, dark urine caused by the release of hemoglobin, and jaundice.

There is no dispute that the current research on arsine supports the route of toxicity outlined above. The parties dispute, instead, whether the conventional toxicological wisdom on arsine exhaustively details the potential for arsine injury. Because many of the plaintiffs in this matter presented with none of the standard signs of arsine exposure, they seek to offer testimony suggesting alternative routes of arsine injury. Defendant, in turn, argues that the pathway of injury outlined in the existing toxicological research encompasses arsine's toxic potential and that individuals whose symptoms do not match that framework may not plausibly claim injury from arsine exposure.

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[4] Defendant contends that arsine injury is impossible without hemolysis; plaintiffs disagree.

Faced with this debate and a voluminous record, the Court must determine whether plaintiffs' experts have developed their opinions, challenging the existing knowledge on arsine toxicity and diagnosing plaintiffs with arsine injury, in a sufficiently reliable manner to allow their admission under Rule 702.

A.[5]

Richard Hastings, D.O. is a doctor of osteopathy in Tulsa, Oklahoma. In addition to his medical degree, he holds a Ph.D. in anatomy and is board certified in internal medicine. Dr. Hastings has no advanced degrees in toxicology or pharmacology and has produced no peer-reviewed literature on the topic of toxicology generally or arsine gas specifically. Of the three plaintiffs' experts, he is the only one who had any contact with plaintiffs, either by phone or in person.[6]

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5 Solkatronic filed a motion to strike (Dkt. # 68) the testimony of Dr. Hastings on the ground that he altered his opinion to include a theory of dual toxicity, which posits a potential exposure to both arsenic and arsine on the date of the release at issue. The Court referred that motion to United States Magistrate Judge Paul J. Cleary, who issued an order on the motion (Dkt. # 82), finding that Dr. Hastings did add to his original Rule 26 report, but could find no reason to strike Dr. Hastings's new opinion, since he developed it on the basis of a review of Rule 26 reports submitted by defense experts Carter and Pike. Accordingly, he denied defendant's motion to strike. Defendant has filed an appeal of Magistrate Judge Cleary's order (Dkt. #85). Having reviewed the magistrate judge's order, the Court cannot find that Judge Cleary made a decision "cleary erroneous or contrary to law," a finding required by Fed. R. Civ. P. 72(a) before this Court will set aside any portion of the magistrate judge's order.

6 Plaintiffs contend that, given the infrequency of human exposure to arsine and the resulting dearth of research on the subject, Hastings's evaluation of most of the plaintiffs in this action makes him one of the foremost authorities on arsine toxicity. Defendant Solkatronic Chemical, Inc.'s Primer on the Science and Medicine of Arsine Poisoning (hereinafter "Defendant's Primer") (Dkt. # 88), Ex. 3A, Deposition of Richard Allen Hastings, II, D.O. (hereinafter "Hastings Deposition"), at 229. This characterization assumes, of course, the fact of plaintiffs' exposure.

When plaintiffs solicited his opinion, Dr. Hastings informed them that he would have to conduct a preliminary literature review to form an opinion as to general causation, that is, whether arsine gas can produce toxic effects when inhaled by humans. Upon completing that review and conducting physical examinations, Dr. Hastings concluded that all of the plaintiffs suffer from arsine-induced injury resulting from the July 11 release. Dr. Hastings produced a written literature review entitled "Historical Scientific and Medical Perspectives Regarding Arsine Gas" (hereinafter "Hastings Literature Review"), in addition to individual reports outlining his findings as to each plaintiff.

The centerpiece of Dr. Hastings's opinions in this case is a general causation theory he calls "biotransformation".[7] That theory builds upon the principle that arsine may transform into a form of arsenic[8] as a result of metabolic processes within the human body and is premised upon a theory of arsenic, rather than arsine, poisoning. The substance of Hastings's biotransformation theory is contained in a paragraph appearing in each of the individual reports submitted in this matter:

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[7] The Court would not normally dwell on the general causation theory underlying an expert's opinion as to a known toxin. See McClain v. Metabolife Int'l, Inc., 401 F.3d 1233, 1239 (11th Cir. 2005) (noting that a "court need not undertake an extensive Daubert analysis on the general toxicity question when the medical community recognizes that the agent causes the type of harm a plaintiff alleges"). Because Dr. Hastings has proposed a previously unrecognized mechanism by which arsine may produce injury, that is, as the result of biotransformation to arsenic in the absence of hemolysis, the Court addresses the reliability of that general causation theory.

[8] There was an extended discussion at the Daubert hearing regarding the many forms of arsenic present in the environment and the imprecision of using the term "arsenic" without specifying to which form one is referring. Since there is no agreement on the forms of arsenic potentially implicated in the present dispute, the Court, for ease of reference, utilizes the term "arsenic" or "a form of arsenic" to refer to any of the forms of arsenic.

> Additional scientific studies have documented the biotransformation of arsine gas within the body to other arsenic forms to include the trivalent and the pentavalent forms. As a group, these various types of arsenic inclusive of the trivalent, pentavalent, and arsine gas have been documented by scientific studies within the body following an acute arsine gas toxic exposure. Many of the direct effects experienced by individuals experiencing an acute toxic arsine gas poisoning result not only from the actions of the arsine gas itself but from the deleterious injury to the body from the biotransformed byproducts that do include the trivalent and pentavalent forms of arsenic.

See, e.g., Defendant Solkatronic Chemical, Inc.'s Motion to Strike Dr. Gad's Second Expert Report and Brief in Support (Dkt. # 52), Ex. 4, Internal Medicine Evaluation for Joshua Wayne Hinton, at 8. Arsenic created within the body may, Dr. Hastings claims, go on to produce any number of harmful effects.

Implicit in Dr. Hastings's biotransformation theory is a proposition from which much of the controversy in this matter grows, namely, his view that an individual exposed to arsine which biotransforms to arsenic internally may suffer injury in the absence of hemolysis. Dr. Hastings hypothesizes that arsine could convert to arsenic before producing a hemolytic effect in the red blood cell. He reasons that because arsenic does not share arsine's high affinity for the red blood cell, the process of biotransformation could occur without the expected hemolytic effects on the system.

Dr. Hastings's theory of biotransformation is, ostensibly, the product of his melding of two scientific principles he attributes to existing published research on the subject of arsine toxicity: first, that arsine converts to arsenic within the body and, second, that arsine may cause injury without hemolysis. The doctor offers a series of articles to support both claims. As to the first principle, Dr. Hastings relies on a 1997 article by Pietro Apostoli and others, entitled Metabolism of Arsenic After Acute Occupational Arsine Intoxication, J. Toxicology & Envtl. Health 331 (1997), as support

for the proposition that arsine may transform to arsenic within the human body. In that article, the authors examine the forms of arsenic resulting from an occupational exposure to arsine gas by examining urine and blood samples from a worker who experienced an acute arsine exposure. During his deposition, Dr. Hastings also cited an article by defense expert Dr. Dean Carter and John B. Sullivan, Jr., entitled Intermetallic Semiconductors & Inorganic Hydrides, Medical Toxicology of Hazardous Substances 916 (John B. Sullivan, ed. 1992), wherein the authors review the toxic effects of various intermetallic semiconductor substrates, including arsine, used in the microelectronics industry. The authors note that chronic low level exposure to arsine could, in some cases, result in higher than usual excretion of urinary arsenic.

Dr. Hastings cited two studies during the Daubert hearing, which he claims substantiate his theory of hemolysis-free arsine injury: a 1991 article by Martin Risk and Laurence Fuortes and a 2000 article by various authors, including defendant's expert, Dr. Carter. In the first article, Chronic Arsenicalism Suspected from Arsine Exposure: A Case Report and Literature Review, Veterinary & Hum. Toxicology 590 (1991), Risk and Fuortes review the clinical history of a 35-year old male who suffered from the effects of a suspected occupational arsine exposure. Among the authors' observations in the piece is the absence of hemolysis in the exposed individual. The authors of the second article, Structural Alterations in the Rat Kidney After Acute Arsine Exposure, Laboratory Investigation 87 (2000), sought to reconstruct the effects of arsine gas on the kidney by exposing renal cortical epithelial cells and a rat kidney to arsine gas. That study documented a direct toxic effect on the kidneys following exposure.

The Court proceeds to its consideration under Daubert of the reliability of Dr. Hastings's biotransformation theory. As a starting point, the Court notes that Dr. Hastings crafted his opinions on the mechanism of arsine injury for the purpose of this litigation.[9] While this does not preclude him from testifying, an expert offering testimony under these circumstances must make an adequate showing as to the validity of the methods undertaken to reach his conclusion.

> That the expert failed to subject his method to peer-review and to develop his opinion outside the litigation is not dispositive, but if these guarantees of reliability are not satisfied, the expert must explain precisely how he went about reaching his conclusions and point to some objective source . . . to show that he has followed the scientific method, as it is practiced by (at least) a recognized minority of scientists in his field.

Estate of Mitchell v. Gencorp, Inc., 968 F. Supp. 592, 600 (D. Kan. 1997) (citing Lust v. Merrell Dow Pharm., Inc., 89 F.3d 594, 597 (9th Cir. 1996)) (internal quotation marks omitted).

Although Dr. Hastings conducted an extensive review of the applicable literature on the subject of arsine toxicity, that review apparently produced no published support for the theory of biotransformation offered by Dr. Hastings. He acknowledged as much during his deposition:

> Q: Is there any medical literature that you have reviewed that discusses the biotransformation of arsine to arsenic in the absence of hemolysis?
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> A: To the best of my recollection, I haven't encountered up to this point in the past literature specific articles that would discuss that transformation process in the absence of hemolysis.

Defendant's Primer, Ex. 3A, Hastings Deposition, at 266.

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[9] Indeed, Dr. Hastings acknowledged at his deposition that he had no knowledge of the toxicological effects of arsine gas prior to engaging in a literature review for the purposes of this case. Defendant's Primer, Ex. 3A, Hastings Deposition, at 64-65.

The Court has independently reviewed the articles cited by Dr. Hastings and finds no support for his theory therein. While the Apostoli article does discuss the conversion of arsine to a form of arsenic, it does not substantiate any claim that arsine converted into arsenic within the body may cause injury without hemolysis or its attendant physical effects. Indeed, the article tends to refute that position, as the individual under evaluation in the study presented with dark red urine, one of the telltale signs of hemolysis. Dr. Hastings admitted that his biotranformation theory departs from the findings of the Apostoli article in this important regard. Id. at 135 ("The reference you cited which is Apostoli, that particular gentleman did have quite a massive hemolysis, and he did have ars[e]nic coming out of the urine.").

When asked, Dr. Hastings noted that he had, likewise, found no literature in support of the underlying premise of his biotransformation theory, that arsine injury is possible without hemolysis.

> Q: Have you seen any medical literature that you have reviewed in formulating your opinions that say you can have a toxicological harmful reaction to arsine gas in the absence of hemolysis?
>
> A: No, I don't recall that I have.

Id. at 232. Indeed, neither of the articles Dr. Hastings cited during the Daubert hearing validates his theory of hemolysis-free arsine injury. The 1991 Risk and Fuortes article, Dr. Hastings conceded during his testimony before the Court, examines a case of chronic, not acute, arsine poisoning, a distinction the authors suggest might explain the subject's lack of hemolysis. Risk & Fuortes, supra, at 595 ("The apparent lack of hemolysis during the course of this toxicity was unusual; this is almost invariably noted in acute arsine poisoning."). Defense expert, Dr. Carter, a co-author of the 2000 article evaluating the effect of arsine on the rat kidney, pointed out during his testimony at the Daubert hearing that the scientists conducting those experiments utilized isolated kidney cells to

force a direct interaction with the kidney. The red blood cell, the site of hemolysis, was excluded for the very purpose of producing that effect. What the results of those tests would have been had the red blood cell been included is both unknown and irrelevant. The article does not bear the significance Dr. Hastings seeks to attribute to it.[10]

Finally, the deposition testimony and written reports of Dr. Hastings reveal that he relied substantially on a Material Data Safety Sheet (MSDS) in educating himself on the effects of arsine exposure. When asked at the Daubert hearing, Dr. Hastings testified that he knew nothing about the source of the information contained in the MSDS, however. When an expert purports to offer an opinion based upon his review of existing literature, it is that critical the proposed expert carefully review the methodology utilized by the scientist conducting the study to ensure the quality of the assumptions and data therein. Mitchell v. Gencorp, Inc., 165 F.3d 778, 783 (10th Cir. 1999). Such a review is impossible when the expert has no knowledge of the procedures followed in acquiring the information contained in the MSDS. In this case, Dr. Hastings had no way to evaluate the quality of the research, if any, underlying the information contained in the MSDS, and that document, as a result, does not bolster his opinion. See Turner v. Iowa Fire Equip. Co., 229 F.3d 1202, 1209 (8th Cir. 2000) (suggesting that expert's ignorance of the tests utilized to formulate MSDS diminished scientific reliability of MSDS); Moore v. Ashland Chem. Inc., 151 F.3d 269, 278 (5th Cir. 1998) (finding that MSDS had limited scientific value to expert when it was unclear what tests were conducted in generating the MSDS).

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[10] Plaintiffs' reliance on Dr. Carter's earlier work is also problematic, since Dr. Carter has disclaimed many of the views once espoused in those pieces in light of his more recent laboratory work.

In sum, the materials cited by Dr. Hastings do not validate his theory of hemolysis-free biotransformation. Although certain articles advance the view that arsine metabolizes into arsenic within the human body, almost all of the articles submitted to the Court identify hemolysis and resulting hemoglobinuria, or blood in the urine, as the paradigmatic signs of arsine gas exposure. See, e.g., Walter T. Klimecki & Dean E. Carter, Arsine Toxicity: Chemical and Mechanistic Implications, J. Toxicology & Envt'l Health, 399, 405 (1995) (recognizing hemolysis and erythroid regeneration as "key indicators of arsine exposure"); Bruce Fowler & Joseph Weissberg, Arsine Poisoning, 291 New Eng. J. Med. 1171, 1171 (1974) ("The most striking and consistent laboratory finding is anemia of a hemolytic type.").

Faced with the absence of unequivocal published support for their expert's theory of biotransformation, plaintiffs emphasize the preliminary nature of the research on arsine. They correctly point out that the absence of scholarly support for a scientific proposition is not necessarily fatal to a proposed expert's ability to offer testimony. See, e.g., Daubert, 509 U.S. at 593 ("Publication . . .is not a sine qua non of admissibility . . . ."); Hollander v. Sandoz Pharm. Corp., 289 F.3d 1193, 1211-12 (10th Cir. 2002)(" We do not believe that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness."). Other circuits have similarly held that Daubert makes room for methodologically sound, albeit unpublished, scientific views. See, e.g., Amorgianos v. National R.R. Passenger Corp., 303 F.3d 256, 266-67 (2d Cir. 2002) ("This is not to suggest that an expert must back his or her opinion with published studies that unequivocally support his or her conclusions . . . Where an expert otherwise reliably utilizes scientific methods to reach a conclusion, lack of textual support may go to the weight, not the admissibility of the expert's testimony.")

(internal quotation marks and citations omitted); Bonner v. ISP Technologies, Inc., 259 F.3d 924, 929 (8th Cir. 2001) ("There is no requirement that a medical expert always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness.") (internal quotation marks and citations omitted); Heller v. Shaw Indus., Inc., 167 F.3d 146, 155 (3d Cir. 1999) ("We do not read the Supreme Court as requiring a medical expert to always rely on published studies indicating the exposure necessary to cause a particular illness.").

While it is not dispositive that Dr. Hastings's biotransformation theory is not confirmed by existing published research on the subject of arsine, its absence from the body of knowledge on arsine toxicity is not without significance. Dr. Hastings, it seems, celebrates the novelty of his theory:

> [T]he results of my findings, which represent the largest population of humans ever studied relative to an acute event, is going to be groundbreaking information scientifically. . . .
>
> And if you go back and look at the old literature, almost every single article that I've seen says, well, we know this much at this point, but there's still something else that needs to be done, there's more data that needs to be obtained. We don't know about this. We don't know about that.
>
> And what I was trying to explain yesterday was that, based upon this large segment of population, it is my opinion that we are evolving science to the point of understanding that not everyone that experiences an arsine exposure toxicity, especially in this particular unique scenario, the combustion explosion thing, has to present with what we used to know in the literature as being established as the classic triad. . . .
>
> And I think — I honestly think that in ten years from now they're going to look back at what we're doing right now and saying, yeah, we thought that everybody had to have the classic triad that included hemoglobinuria for an arsine poisoning, but as a result of the evolution of the science based upon this large a study in human mankind doctoring, we realize now that there is a gradient.

> There is arsine toxicity with illness in chronic arsenicals that doesn't necessarily have to result in the presentation of the classic triad. And that's--that's what I was trying to get across to you. It goes to the comparison of a single patient in one study to a massive group of patients.
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> And I don't think that you can extrapolate directly based on the previous scientific information that's been presented that did encompass smaller groups to this particular larger group that has the eccentricities of this exposure.

Defendant's Primer, Ex. 3A, Hastings Deposition, at 230-32; id. at 244.

In the absence of published support, however, the Court must find other indicators of reliability, and, in this regard, the manner by which Dr. Hastings developed his theory of biotransformation takes on particular importance. For this Court, Daubert's emphasis on the utility of testing as a measure of scientific reliability becomes critical. Daubert, 509 U.S. at 593 (identifying as a "key question" whether a theory can be or has been tested). The law of this circuit does not require that an expert who propounds an unpublished theory of causation always conduct testing prior to gaining admission of that theory. See, e.g., Bitler, 400 F.3d at 1236 (holding that testing was not necessary when reliability of the science of copper sulfide contamination was not in dispute). However, "[w]hen an expert proposes a theory that modifies otherwise well-established knowledge about regularly occurring phenomenon . . . we would expect the importance of testing as a factor in determining reliability to be at its highest." Id. at 1235-36.

Dr. Hastings's biotransformation theory has been the subject of no scientific testing and study, however.[11] The theory is the product of the literature review Dr. Hastings conducted in

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[11] Plaintiffs insist that it would be impossible to test his theory, because no scientist could ethically expose human subjects to arsine for the sole purpose of evaluating the accuracy of a theory of toxicity. Plaintiffs' insistence that testing is an impossibility is belied by the existence of research on arsine's toxicity. In any event, as the Court explains, Dr. Hastings has also failed to provide an analytically precise explanation of the process of biotransformation.

preparation for this litigation. From what is known--that arsine may convert to arsenic within the human body--Dr. Hastings inferred that the transformation may occur, first, without the arsine binding to and prompting the lysis of the red blood cell, and, second, in a manner that permits the converted arsenic to go on to produce permanent physical damage in an exposed individual. Dr. Hastings has, apparently, made no effort to test the accuracy of this inference, however.

Having failed to complete any tests validating his theory of biotransformation, Dr. Hastings offers little more than conjecture on the process by which this hemolysis-free conversion occurs. Dr. Hastings's own testimony is instructive in this regard. Asked to explain the mechanics of his biotransformation theory, Dr. Hastings provided speculation:

> Q: Well, how does the mechanism of your biotransformation theory work? How does the arsine biotransform to arsenic?
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> A: Well, it happens through a chemical process during the metabolism within the body fluids or within the cells that it injures or the cells that are metabolized by it.
>
> Q: Okay. And how does happen?
>
> A: I don't know that I can give you the specific mechanism at this point as to how that happens. There are a lot of postulated mechanisms for various types of reactions and ---but I'm not sure that I'm able to do that.
>
> What I can tell you is that in these individuals that experience an arsine exposure, arsine goes in and arsenic comes out, and so that should be an indication that there is a process that happens. I don't know the exact chemistry. I'm not sure that anyone is exactly sure of all those events, but certainly I don't know the exactly chemistry of that mechanism.

Defendant's Primer, Ex. 3A, Hastings Deposition, at 273. Indeed, at one point, Dr. Hastings recognized that his theory might be incorrect.

> Q: So, it's possible in this case, Doctor, that these plaintiffs could have inhaled arsine, and excreted out through their urine an arsenic byproduct that wouldn't have caused them any physical harm at all? Is that possible?
>
> A: I can't say that's impossible, yeah. That's a possibility.

Id. at 283.

While the primary concern of this Court is not with the accuracy of Dr. Hastings's conclusions, Daubert, 509 U.S. at 595, the Supreme Court has made clear that an expert's conclusions may be the subject of particular scrutiny when they move beyond the boundaries of rational inference into the realm of speculation. As the Court explained,

> Trained experts commonly extrapolate from existing data. But nothing in either Daubert or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the ipse dixit of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered.

General Elec. Co. v. Joiner, 522 U.S. 136, 146 (1997); see also Bitler, 400 F.3d at 1233 ("[W]hen the conclusion simply does not follow from the data, a district court is free to determine that an impermissible analytical gap exists between premises and conclusion.").

The Court so concludes as to Dr. Hastings's theory of biotransformation. Without question, Dr. Hastings considers his views on the mechanism of arsine poisoning to be at the forefront of thinking on the subject of arsine toxicity. There is no evidence in the record, however, that the biotransformation theory is either supported by existing research on the topic of arsine or is the product of testing conducted by Dr. Hastings. Moreover, Dr. Hastings's own analysis on the theory of biotranformation suffers from inexcusable analytical gaps. See Amorgianos, 303 F.3d at 267 ("To warrant admissibility, . . . it is critical that an expert's analysis be reliable at every step. . . .").

This Court may not, consistent with the mandate of Daubert and its gatekeeping function, permit this litigation to become a forum in which an expert may offer what is, presently, mere speculation on the possibilities of alternate routes of arsine injury. "[T]he courtroom is not the place for scientific guesswork, even of the inspired sort. Law lags science; it does not lead it." Rosen v. Ciba-Geigy Corp., 78 F.3d 316, 319 (7th Cir. 1996). While Dr. Hastings may well stand on the precipice of a scientific breakthrough, he has not adequately established that his biotransformation theory is the product of sufficiently rigorous scientific inquiry or a grounding in the methods of science. The Court in Daubert, noting the "important differences between the quest for truth in the courtroom and the quest for truth in the laboratory," acknowledged that the reliability mandate embodied in Rule 702 may exclude from the courtroom hypothetical musings that would be embraced in the laboratory. As the Court explained,

> Scientific conclusions are subject to perpetual revision. Law, on the other hand, must resolve disputes finally and quickly. We recognize that, in practice, a gatekeeping role for the judge, no matter how flexible, inevitably on occasion will prevent the jury from learning of authentic insights and innovations. That, nevertheless, is the balance that is struck by Rules of Evidence designed not for the exhaustive search for cosmic understanding but for the particularized resolution of legal disputes.

Daubert, 509 U.S. at 596-97. The Court, therefore, will not permit Dr. Hastings to testify regarding his biotransformation theory of arsine exposure. See Truck Ins. Exch. v. Magnetek, Inc., 360 F.3d 1206,1213 (10th Cir. 2004) (affirming district court's exclusion of expert who proposed theory of

pyrolosis because the theory was "not yet a sufficiently reliable scientific theory upon which to base an expert opinion" as to causation).[12]

In addition to his biotransformation theory of exposure, Dr. Hastings submits a theory of dual toxicity, in which he posits that plaintiffs may have inhaled arsenic, in addition to arsine, on the date of the release. Although Dr. Hastings originally rejected the possibility that plaintiffs inhaled arsenic, he subsequently altered his opinion to include the possibility that the exothermic reaction produced by the explosion of the canister containing the arsine gas may have generated arsenic that was later inhaled by plaintiffs. This theory was not explored in any of Dr. Hastings's written reports, but he provided a brief explanation during his deposition:

> I am adding to my medical opinion that I think that there was more than one toxicant expressed at the scene of the explosion . . . [F]rankly [,] the toxic effects of ars[e]nic would fit a majority of these patients' clinical conditions that don't involve hemolysis more than directly, solely being exposed to arsine.

Defendant's Primer, Ex. 3A, Hastings Deposition, at 141. Dr. Hastings generated his new opinion after a review of the Rule 26 reports of Dr. Carter and Dr. Pike. Id. at 144.

Dr. Hastings's theory of dual toxicity suffers from similar methodological problems, though these problems are most apparent in the application of the theory to the individual plaintiffs. While the idea that arsine may convert to arsenic as a result of combustion is largely uncontested, Dr. Hastings's opinions as to the individual plaintiffs' exposure to arsenic flies in the face of existing information on the July 11 event.

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[12] Because the Court concludes that Dr. Hastings's biotransformation theory is not a reliably developed theory of general causation, it does not consider its application to the individual plaintiffs, for "without general causation, there can be no specific causation." Norris, 397 F.3d at 881.

Dr. Hastings claims that plaintiffs may have inhaled arsenic as well as arsine following the July 11 release. At his deposition, however, Dr. Hastings acknowledged that the result of soil sampling conducted at the site[13] confirmed that there had been no impact to the soil from the July 11 event, undermining any suggestion that any released arsenic escaped the Solkatronics facility to be inhaled by plaintiffs. Defendant's Primer, Ex. 3A, Hastings Deposition, at 103. Dr. Hastings rejected the results of that testing, alleging that the engineers had relied upon inaccurate meteorological data in reaching their results. Id. While Dr. Hastings may reasonably take issue with both the methods and the results of the testers, he neither offered alternative results upon which he relied in drawing his causation opinions, nor made an independent attempt to evaluate arsenic concentration levels in the affected area. Id. at Ex. 3B, Hastings Deposition, at 392-93. Further, although Dr. Hastings claims that certain plaintiffs may have inhaled some form of arsenic, a solid, he agreed at his deposition that none of the plaintiffs recalled encountering a particulate on the day of the release. Id. at Ex. 3A, Hastings Deposition, at 160. Having rejected the validity of the only existing soil sampling results and having neglected to do any independent testing, the Court fails to understand how Dr. Hastings could reliably draw any conclusions as to plaintiffs' environmental

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[13] Soil testing was completed through the collection and analysis of soil samples from both upwind and downwind of the release. Four soil samples were collected from the downwind portion of the site and two samples from the upwind portion to represent background concentrations of arsenic in the area. Each sample was split into three parts, for a total of eighteen soil samples. Statistical analysis of the samples from upwind and downwind found no statistical difference in the arsenic concentrations of the respective samples. Engineers concluded on the basis of the testing that there had been no impact to the soil--that is no increase in arsenic concentration in the soil--as a result of the July 11 release. Defendant's Primer, Ex. 25, Soil Sampling Results at Solkatronics Facility, Catoosa, Oklahoma.

exposure to arsenic. The Court determines, therefore, that Dr. Hastings's specific causation opinions based on his dual toxicity theory may not be admitted.[14]

Even were the Court to overlook the other apparent failings of Dr. Hastings's diagnostic process, there is an additional, significant, impediment to the admission of his specific causation opinions. A plaintiff seeking to succeed in a toxic tort case must demonstrate that he or she experienced exposure at a rate sufficient to cause injury. The Tenth Circuit has elaborated on this principle:

> It is well established that a plaintiff in a toxic tort case must prove that he or she was exposed to and injured by a harmful substance manufactured by the defendant. . . . In order to carry this burden, a plaintiff must demonstrate the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover.

Mitchell, 165 F.3d at 781. The methods used to prove the level of exposure must meet a minimal standard of scientific reliability through the use of techniques "subject to objective, independent validation in the scientific community." Id. Expert testimony on the subject of exposure should include description of the method used to arrive at the level of exposure and scientific data supporting the determination. "The expert's assurance that the methodology and supporting data is reliable will not suffice." Id.

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[14] Dr. Hastings also attributed to the hemolytic process the abnormal bloodwork of plaintiffs who presented with heightened plasma free hemoglobin. He noted, however, that none of the plaintiffs's laboratory data confirmed the existence of a hemolytic anemia and concluded that the real agent of the ailments presently complained of was arsenic, not arsine. Defendant's Primer, Ex. 3A, Hastings Deposition, at 239.

Dr. Hastings provided no evidence of either the dose of arsine allegedly inhaled by plaintiffs or any reasonable hypothesis as to the reaction to be expected based on a given dose.

> Q: Now, with regard to all of the plaintiffs, do you have any opinion as to the level of toxicant that any of these people may have been exposed to?
>
> A: No. I don't have any specific information as to the level of toxin. . . . We determined that it was not even sure how much of the toxicant was released so that I don't have a specific number that I can put on the level of the toxicant to which they were exposed.

Defendant's Primer, Ex. 3B, Hastings Deposition, at 448. This ignorance extends to Dr. Hastings's theory of dual toxicity. Id. at 152. Without such information, Dr. Hasting's causation theories as to the individual plaintiffs amount to mere guesswork. Mitchell, 165 F.3d at 781. Guesses, even if educated, are insufficient, however, to merit admission under Rule 702. Id.

Finally, the Court notes what it considers a fundamental problem in Dr. Hastings's opinions in this case. Dr. Hastings posits two theories by which plaintiffs may have been injured--biotransformation or dual toxicity. Having offered these two theories of exposure, Dr. Hastings's written reports provide no insight into where in his taxonomy of exposure scenarios each plaintiff falls. Each written report goes no further than to find that within a reasonable degree of medical certainty, a plaintiff's exposure to arsine gas caused his or her medical symptoms. See, e.g., Internal Medicine Evaluation for Doug M. Ingram, at 8. The reports do not state whether a given plaintiff suffers from poisoning from arsenic biotransformed from arsine within the body or the inhalation of a combination of both arsine and arsenic on the date of the release.[15] Given the differences between arsine and other arsenicals in chemical makeup and toxicity alone, one would expect such

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[15] After Dr. Hastings offered his new theory of causation--dual toxicity--he did not apprise the parties or the Court of which plaintiffs he believes suffered from the inhalation of both arsine and arsenic on the date of the release.

a distinction to be critical in formulating specific causation opinions based on a given patient's symptoms. While this omission does not, standing alone, exclude Dr. Hastings as an expert in this case, it reflects, in the Court's view, a certain lack of scientific precision in the development of Dr. Hastings's opinions.

As the proponents of their experts' opinions, plaintiffs bear the burden of demonstrating the admissibility of each expert's testimony. Ralston, 275 F.3d at 970 n.4. The Court holds that plaintiffs have not met this burden as to Dr. Hastings. In so holding, the Court recognizes that Daubert "neither requires nor empowers trial courts to determine which of several competing scientific theories has the best provenance." United States v. Mitchell, 365 F.3d 215, 244 (3d Cir. 2004). All that is required is that the proponent of the evidence demonstrate that the proposed expert reached his conclusions in "a scientifically sound and methodologically reliable fashion." Id. Because the Court concludes that Dr. Hastings's opinions as to his theory of biotransformation lack a grounding in the methods of science and that his remaining specific causation opinions suffer from various methodological shortcomings, it declines to admit Dr. Hastings's causation opinions.

Dr. Hastings, if he is to testify, is restricted to the symptoms reported by plaintiffs to him and his objective findings when examining plaintiffs. He may not express an opinion on the cause of those diagnoses, having reached his conclusions in an impermissibly unreliable fashion.

B.

Robert Harrison, M.D., is a professor of medicine at the University of California-San Francisco. He holds a master's degree in public health and has authored or co-authored numerous articles in peer-reviewed journals. He has particular expertise in the area of occupational injury and provides consultation to the state of California in workmen's compensation actions filed within

the state. Dr. Harrison became involved in this litigation approximately three years after the July 11 release and never met with, or personally examined, any of the plaintiffs. Instead, he relied upon medical reports made available to him by plaintiffs and the written reports prepared by Dr. Hastings.

Unlike Dr. Hastings, however, Dr. Harrison does not take issue with the prevailing opinion that hemolysis is the principal injury caused by arsine exposure. Indeed, at his deposition, he rejected the view that arsine injury is possible absent lysis of the red blood cell:

> Q: And could they hypothetically have health effects without evidence of hemolysis?
>
> A: The individuals could have psychological effects as a result of the exposure, but I doubt whether they could have physical effects without evidence of hemolysis.

Defendant's Primer, Ex. 1A, Deposition of Robert Jay Harrison, M.D., M.P.H. (hereinafter "Harrison Deposition"), at 118. Instead, Dr. Harrison's specific causation theories are based upon his belief that an accurate diagnosis of hemolysis and arsine injury may be made in the presence of only one abnormal blood reading. Id. at 211. In this case, Dr. Harrison relied upon heightened plasma free hemoglobin, conceding that lab data acquired immediately after the July 11 release[16] revealed none of the other indicators of hemolysis, including decreased haptoblobin, hemoglobin, or hematocrit. Id. at 187.

Dr. Harrison's written report does not distinguish among the plaintiffs. It merely provides that each plaintiff demonstrated "symptoms of acute arsine intoxication immediately following the July 11, 2001 incident," including headache, a metallic taste, nausea, numbness, tingling in the extremities, and shortness of breath. Report of Robert Harrison, at 4. Dr. Harrison's report

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[16] Five plaintiffs--Biddle, Cardenas, Castro, Guerra, and Schnitzer--were not seen on July 11, 2002, and therefore, no lab data immediately following the release exist for those plaintiffs.

continues, "All [thirteen] clients have experienced either central or peripheral nervous system effects subsequent to the acute arsine intoxication." Id. It concludes,

> [T]he July 11, 2001 arsine gas release resulted in acute arsine intoxication among many individuals in the proximity of the Solkatronics Chemical facility. The acute symptoms reported by the [thirteen] plaintiffs are consistent with the known health effects of arsine reported in the medical and scientific literature following acute intoxication. Furthermore, these individuals continue to have chronic neurological problems that are probably caused by the arsine gas exposure. The complete extent of the neurological damage may be assessed by complete neuropsychological evaluation and testing of peripheral nerve function.

Id. at 5.

Although Dr. Harrison's report suggests that he believes that each plaintiff suffers from arsine-induced injuries, during his deposition, he assigned the symptoms of only Javier Cardenas, Linda Castro, Teresa Haggard, Josh Hinton, Doug Ingram, Allan Miller, Dale Patton, Bart Schnitzer, and Joe Sumter to arsine exposure on July 11, 2001.[17]

Defendant objects to the view advanced by Dr. Harrison that an accurate diagnosis of arsine toxicity may be made in the presence of one abnormal lab result, in this case, heightened plasma free hemoglobin readings. In particular, defendant attacks the methodology utilized by Dr. Harrison in arriving at his specific causation opinions as to each plaintiff.

Dr. Harrison testified that he reached his causation opinions after completing a differential diagnosis. "Differential diagnosis refers to the process by which a physician rules in all scientifically plausible causes of the plaintiff's injury. The physician then rules out the least

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[17] During his testimony, Dr. Harrison agreed with the conclusions of Dr. Gad's 2004 report that insufficient information existed to draw any causation conclusions as to plaintiffs Biddle, Guerra, and Shavers and declined to attribute plaintiff Schnitzer's renal failure to exposure to arsine gas on July 11, 2001. During the course of his deposition, he withdrew his opinions as to Joshua Hinton.

plausible causes of injury until the most likely cause remains. The remaining cause is the expert's conclusion." Hollander., 289 F.3d at 1209; see also Kannakeril v. Terminix Int'l, Inc., 128 F.3d 802, 807 (3d Cir. 1997) ("Differential diagnosis is defined for physicians as the determination of which of two or more diseases with similar symptoms is the one from which the patient is suffering, by a systematic comparison and contrasting of the clinical findings.") (internal quotation marks omitted). The Tenth Circuit has held that a properly executed differential diagnosis may be sufficiently reliable to garner admission under Rule 702. See, e.g., Goebel v. Denver & Rio Grande Western R.R. Co., 346 F.3d 987, 998 (10th Cir. 2003).

During his deposition, Dr. Harrison outlined his standard diagnostic protocol when called upon to evaluate the cause of a given physical ailment:

> I take a history . . . which includes details about the exposure, what do we know about the chemical that was used, how was it released into the air, what was somebody doing at the time of exposure, something about the ventilation, if they're directly working with a chemical that applies, probably in this case it's not pertinent. Were there--was there a respirator or mask or some other protective device that was used that might have decreased the exposure, were they inside or outside, what was the time of the release, and when was their--when did their symptoms first start. . .
>
> I want to know something about your previous medical history, because I want to know if there's been a change of your symptoms. I want to know about other medical conditions, medications you take, alcohol you use.
>
> I want to know, then, did you go to get treatment. . . . I'll go through your previous occupational history. I want to know what you did in the past, did you have chemical exposures or some other exposures, you know, that built up that might affect your health now.
>
> I will then do a physical examination, see if there's any objective signs of health effects that is often focused on your complaints or what I know about the health effects of the chemicals to which you have been exposed.
>
> I then might order tests that try to confirm my suspicion. There's a whole host of different kinds of tests depending on what the chemical is. . . . I'd then get your medical records to see what kind of treatment you had. Not everybody can

> remember everything for which they had treatment. I'll fill in the gaps by looking at the medical records. And that helps me evaluate the credibility, which is also an important consideration. In my field if somebody has exposure and they're involved in litigation, I always consider whether or not somebody has secondary gain, whether they may be exaggerating their complaints.

Defendant's Primer, Ex. 1B, Harrison Deposition, at 224-28.

In material respects, Dr. Harrison's approach to diagnosing plaintiffs in the instant case departed from the protocol outlined above. He did not personally examine any of the plaintiffs or conduct his own testing. Rather, he relied on existing medical records and the conclusions of Dr. Hastings in reaching his opinions. Dr. Harrison's reliance on Dr. Hastings's findings, does not, as a matter of law, completely undercut his ability to offer an opinion as to specific causation. See Kannakeril, 128 F.3d at 807 ("Depending on the medical condition at issue and on the clinical information already available, a physician may reach a reliable differential diagnosis without himself performing a physical examination."). The Court's findings as to the reliability of Dr. Hastings's diagnoses does undermine, to some extent, Dr. Harrison's conclusions, however.

Moreover, Dr. Harrison lacked a complete medical and occupational history for each of the plaintiffs. He testified:

> Q: When you say, "Their medical records," could you tell me without obviously going through each individual person that you were asked to review the general nature of the records that you received. And by the general nature I mean the time span that they covered.
>
> A: The medical records generally covered the date of the incident or the few days after the July 11, 2001 incident and follow-up medical examinations. On some of the individuals there were medical records prior to the July 11th, 200[1] date. I don't believe that there were comprehensive medical records on all of them, but generally speaking, I was satisfied that there was medical records that gave me enough information to determine whether or not there were preexisting problems.

Defendant's Primer, Ex. 1A, Harrison Deposition, at 119-20. In some cases, however, Dr. Harrison diagnosed plaintiffs without the aid of medical history he admitted might have altered his conclusions. Certain examples illustrate the point.

Plaintiff Doug Ingram's present complaints include migraine headaches, sleeplessness, numbness, tingling in his extremities, and generalized muscle soreness. Because Dr. Harrison did not consult Ingram's deposition, he was unaware that Ingram had reported to the hospital in December 2000, approximately six months before the release, complaining of severe headaches that had lasted approximately four to six months. Defendant's Primer, Ex. 13, Deposition of Doug Ingram, at 160-61. At that same visit, Ingram reported back pain. Id. Ingram's deposition also suggests that his cholesterol was abnormal for his age and weight prior to the July 11 event. Id. at 154. Dr. Harrison acknowledged that additional information regarding Ingram's previous complaints might have changed his conclusion. Defendant's Primer, Ex. 1B, Harrison Deposition, at 301 ("You know, if there's more information and more detail about his previous medical condition that there hasn't been any change [in the severity of his headaches], my opinion would be altered. I don't have that information.").

Joshua Hinton alleges that due to his exposure to arsine, he now experiences recurrent headaches, chronic fatigue, shortness of breath, and memory loss. Dr. Harrison testified that he relied upon Hinton's plasma free hemoglobin reading, which was one-tenth of a percent above normal, and Hinton's reported symptoms of headache, fatique, shortness of breath, and chest tightness to conclude that Hinton had suffered arsine exposure and injury. At Harrison's deposition, defense counsel questioned Dr. Harrison regarding medical records from the date of the release suggesting that Hinton woke up on July 11 with symptoms and may not have been in the area at the

time of the release. Apprised of that information, Dr. Harrison, without explanation, withdrew his causation opinion as to Joshua Hinton. Id. at 259-60 (Q: Do you believe that Mr. Hinton has any continuing injury as a result of what you've described as exposure to arsine? A: I don't have enough information to offer an opinion.)

Finally, Dr. Harrison relied heavily on plaintiffs' reported symptoms upon arrival at the hospital on July 11--including headache, nausea, and dizziness--to arrive at the conclusion that certain plaintiffs were exposed to arsine gas. It is not clear, however, that he adequately controlled for the possibility that the explanation for plaintiffs' complaints was their extended time in the sun following evacuation on July 11. Doug Ingram testified that on the day of the release, the temperature at the Port of Catoosa exceeded one hundred degrees. Defendant's Primer, Ex. 13, Deposition of Doug Ingram, at 106. When asked, Dr. Harrison agreed that after standing in such temperatures without water, plaintiffs' symptoms may have been the result of heat stress and dehydration, not arsine poisoning. Id. at Ex. 1A, Harrison Deposition, at 121-22. While Dr. Harrison recognized that he would have to know the prevailing weather conditions to control adequately for the possibility of heat stress, id. at 122 ("To the extent that I think that heat stress might be a cause of those symptoms, it would be important to know something about the temperature and humidity at that point in time."), he had no knowledge of those conditions when he made his diagnoses. Id. at Ex. 1B, Harrison Deposition, at 205-06. Thus, Dr. Harrison could not have reliably excluded heat stress as a cause of plaintiffs' earliest reported symptoms, since he was unaware of the temperature on the day of the release or the duration of each plaintiffs' time outside following evacuation. Id. at 205.

A reliable differential diagnosis must, at minimum, provide a reasonable explanation for why an expert excluded a potential alternative cause of a plaintiff's complaint. See Norris, 397 F.3d at 885 ("[T]he expert must 'rule in' the suspected cause as well as 'rule out' other possible causes."). Confronted with alternative explanations of plaintiffs' ailments, Dr. Harrison failed to explain how his diagnoses continued to hold up in the face of additional, and potentially confounding, information. See Kannankeril, 128 F.3d at 808 (noting that when challenging the validity of the differential diagnosis conducted by a proposed expert, "the defendant may point to a plausible cause of the plaintiff's illness other than the defendant's actions. It then becomes necessary for the plaintiff to offer a good explanation as to why his or her conclusion remains reliable."). Indeed, in certain cases, the introduction of new information sufficiently undercut Dr. Harrison's confidence in his ability to draw a reliable conclusion that he withdrew his causation opinion entirely.

The Court again notes that it is not charged with evaluating the accuracy of Dr. Harrison's conclusions. Instead, the Court looks for the hallmarks of reliable, scientific inquiry in reaching those conclusions, in this case, the completion of a properly conducted differential diagnosis. The Court determines that Dr. Harrison lacked the necessary information to differentiate reliably among the myriad potential causes of plaintiffs' complaints. Morever, when provided with information highlighting other possibilities, Dr. Harrison did not offer a plausible explanation of how he ruled out the proffered alternative and ruled in arsine injury. Dr. Harrison's procedures in this matter also appear to depart from his own established diagnostic standards. See Amorgianos, 303 F.3d at 268-69 (affirming district court's exclusion of expert opinion when proposed expert neglected to apply his own methodology reliably). Accordingly, the Court excludes Dr. Harrison's causation

testimony as to plaintiffs on the ground that he conducted a differential diagnosis without the information necessary to exclude reliably alternative sources of plaintiffs' present illness.

C.

Shayne C. Gad, Ph.D. is a board certified toxicologist and a member of the Academy of Toxicologic Science (ATS). He operates Gad Consulting Services, which conducts clinical trials for pharmaceutical companies and provides consultation in litigation matters.

Dr. Gad's opinions in this matter are the subject of a vigorous discovery dispute, and the Court necessarily begins there. Dr. Gad has submitted two written reports: dated December 30, 2004 ("the 2004 report") and April 15, 2005 ("the 2005 report"). The 2004 report provides, in outline form, the established signs and symptoms of arsine exposure and then provides specific causation opinions as to each individual plaintiff. Dr. Gad's 2005 report is a much more detailed review of the science of arsine toxicity and includes an assessment of the opinions offered by defense experts Dr. Dean Carter and Dr. Steven Pike. Dr. Gad criticizes Dr. Carter and Dr. Pike for, inter alia, neglecting to address the possibility that repeated, chronic exposure to arsine, as opposed to a single, acute exposure in July 2001, caused plaintiffs' health problems. Although it is not contained in his original report, at the Daubert hearing, Dr. Gad testified that arsine injury may result without the occurrence of hemolysis by way of direct organ toxicity.

Defendant contends that Dr. Gad should be precluded from offering any of the opinions introduced subsequent to the submission of his initial 2004 report. It has filed a motion to strike his chronic exposure theory of causation, arguing that permitting the introduction of Dr. Gad's chronic exposure theory would irreparably harm defendant. In a brief filed following the Daubert hearing, defendant simultaneously renews and broadens its earlier motion, insisting that the Court should

strike the entirety of the general causation opinions contained in Dr. Gad's 2005 opinion, since they constitute previously undisclosed expert testimony.

The parties' dispute implicates Rules 26 and 37 of the Federal Rules of Civil Procedure. Rule 26 commands that expert reports contain

> a complete statement of all opinions to be expressed and the basis and reasons therefor; the data or other information considered by the witness in forming the opinions; any exhibits to be used as a summary of or support for the opinions; the qualifications of the witness, including a list of all publications authored by the witness within the preceding ten years; the compensation to be paid for the study and testimony; and a listing of any other cases in which the witness has testified as an expert at trial or by deposition within the preceding four years.

Fed. R. Civ. P. 26(a)(2)(B). The rule governing the disclosure of expert opinions serves an important purpose in apprising a party of the views of the opposing party's experts and permits adequate preparation for depositions and cross-examination at trial. See Jacobsen v. Deseret Book Co., 287 F.3d 936, 953 (10th Cir. 2002).

Rule 37 contains a self-executing exclusion mechanism which precludes the use at trial of previously undisclosed expert opinion.

> A party that without substantial justification fails to disclose information required by Rule 26(a) or 26(e)(1) . . . is not, unless such failure is harmless, permitted to use as evidence at a trial, at a hearing, or on a motion any witness or information not so disclosed.

Fed. R. Civ. P. 37(c)(1). "[T]he combined effect of Rule 26(a)(2)(B) and 37(c)(1) is that he who fails to provide a comprehensive expert report does so at his peril." Anderson v. Hale, No. CIV-02-0113-F, 2002 WL 32026151, at *2 (W.D. Okla. Nov. 4, 2002).

In determining whether an expert's failure to disclose testimony constitutes a harmless omission under Rule 37, the Court considers the following factors: (1) the prejudice or surprise to the party against whom the testimony is offered; (2) the ability of the party to cure the prejudice; (3)

the extent to which introducing such testimony would disrupt the trial; and (4) the moving party's bad faith or willfulness. Jacobsen, 287 F.3d at 953; Woodworker's Supply, Inc. v. Principal Mut. Life Ins. Co., 170 F.3d 985, 993 (10th Cir. 1999).

Having compared Dr. Gad's 2004 and 2005 reports, the Court concludes, first, that Gad's chronic exposure theory is not mentioned in the 2004 report and that Dr. Gad's theory of chronic exposure is, indeed, a previously undisclosed theory. The Court further holds that the chronic exposure theory does not constitute evidence "intended solely to contradict or rebut evidence on the same subject matter identified by another party" under Rule 26(a)(2). The Rule 26 reports of Dr. Carter and Dr. Pike contain no reference to a theory of chronic arsine exposure. Dr. Gad's chronic exposure theory may also not properly be characterized as a "supplement" to his 2004 report as it does not amend "incomplete or incorrect" information contained in that report. Fed. R. Civ. P. 26(e) advisory committee's note.

Plaintiffs argue, however, that because defendant had an opportunity to depose Dr. Gad following the submission of the 2005 report, defendant suffered no prejudice as a result of Dr. Gad's inclusion of a chronic exposure theory. Although defense counsel had the opportunity to depose Dr. Gad following the submission of his second report, defendant's experts--the individuals best equipped to evaluate the substance of Dr. Gad's opinions--have not had an opportunity to evaluate with care Dr. Gad's chronic exposure theory and provide formal opinions on the subject. Since Dr.

Pike and Dr. Carter's expertise makes them the individuals best able to appraise the validity of Dr. Gad's views, the Court finds that this inability places defendant at a significant disadvantage.[18]

Additionally, defendant is correct that if the Court were to admit Dr. Gad's chronic exposure theory and defendant wished to have its experts refute that theory at trial, they would be required to testify to opinions not expressed in their Rule 26 reports in violation of Rule 26, since both experts responded exclusively to plaintiffs' acute arsine exposure theory. The Court will not sanction such a violation of the federal rules to accommodate the admission of a heretofore largely unexplored causation theory.

As to the second factor--the ability of plaintiffs to cure the prejudice alleged--the Court determines that plaintiffs are in no position to correct the most serious prejudice visited on defendant, namely, its inability to have its experts conduct a complete review of the chronic exposure theory advanced by Dr. Gad in his 2005 report. The only solution readily apparent to the Court would be re-opening discovery to permit defendant's experts to conduct a full evaluation of the merits of Dr. Gad's theory and produce rebuttal reports, if necessary. This, the Court is unwilling to do at this late date.

These discovery concerns lead to the Court's conclusion as to the third factor. Although permitting the admission of Dr. Gad's new theory would not necessarily disrupt trial, it would upset the progression of this case, which has been pending before the Court for approximately two years.

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[18] In any event, Dr. Gad testified at his deposition that he had not been asked to evaluate plaintiffs for potential chronic exposures and had not made such an evaluation. See, e.g., Defendant's Primer, Ex. 2, Deposition of Shayne C. Gad, Ph.D. (hereinafter "Gad Deposition"), at 248 (Shavers); id. at 305 (Ingram). Whatever his views on the possibility for chronic exposure, Dr. Gad's testimony at his deposition makes clear he has no plans to offer an opinion at trial that any of the plaintiffs suffered a chronic exposure to arsine.

Allowing what could be extensive further discovery on the question of Dr. Gad's chronic exposure theory would further delay whatever issues remain to be litigated in this matter. Finally, as to the question of bad faith or willfulness, the fourth factors, the Court finds no evidence of either. Good faith alone in failing to disclose cannot preserve Dr. Gad's chronic exposure opinion, however. See Jacobsen, 287 F.3d at 954 ("[G]ood faith alone would not be enough to overcome the other factors.").

Accordingly, the Court concludes that, in light of the potential prejudice threatened by the admission of Dr. Gad's chronic exposure theory and the special measures necessary to eliminate that prejudice, the Court strikes Dr. Gad's 2005 report, insofar as it introduces a chronic exposure theory of arsine poisoning. For the purposes of its Daubert determinations, the Court will not consider Dr. Gad's opinion relating to chronic exposure to arsine gas.

The Court reaches a different conclusion as to the remaining general causation opinions expressed in Dr. Gad's 2005 report, including his views regarding the site of metabolism, the potential for delay preceding hemolysis, and the opportunity for direct organ injury from arsine exposure without evidence of hemolysis. "Rule 37(c) permits a district court to refuse to strike expert reports and allow expert testimony even when the expert report violates Rule 26(a) if the violation is justified or harmless." Id. at 952. Defendant's experts have considered and written at length on the remaining general causation opinions contained in Dr. Gad's 2005 report, making a rebuttal of those opinions permissible under Rule 26(a)(2)(C). Although plaintiffs have failed to explain the curious evolution of Dr. Gad's opinions in this matter, admission of his testimony inflicts no readily observable harm on defendant and is contemplated by Rule 26.

The Court turns, then, to the substance of Dr. Gad's opinions. Plaintiffs' counsel represented at the Daubert hearing that plaintiffs seek to offer Dr. Gad as an expert on general causation issues only.[19] During his testimony, Dr. Gad outlined his general causation opinions as follows: (1) Arsine can cause serious injury; (2) You may have injury in the absence of observed hemolytic effects; and (3) The health effects on the human kidney and liver are well-documented. Defendant takes issue with the second proposition, namely, that arsine injury may occur in the absence of hemolysis.

When asked at the Daubert hearing about the basis for his second opinion, Dr. Gad cited two documents: a 2003 article by E. Martin Caravati, Arsenic & Arsine Gas, Medical Toxicology (Dart 3d ed. 2003), and the Medical Management Guidelines (MMGs) for Arsine promulgated by the Agency for Toxic Substances and Diseases Registry (ATSDR), which is affiliated with the Centers for Disease Control and Prevention. The Caravati article examines the chemical makeup, toxicity, clinical presentation, and treatment of both acute and chronic exposure to arsine and various arsenic compounds. While the article places special emphasis on the hemolytic effects of arsine on the human body, including the adverse effects of the hemolytic process on the kidney, it also underscores the possibility of a direct toxic effect on the kidney. The MMGs for arsine, likewise, note that although hemoglobin released into the blood stream and processed through the kidneys is considered the primary source of kidney damage following arsine exposure, "a direct toxic effect of arsine or deposition of the arsine-hemoglobin-haptoglobin complex may also play a role." Defendant's Primer, Ex. 27A, Medical Management Guidelines (MMGs) for Arsine.

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[19] At the Daubert hearing, Dr. Gad disclaimed his specific causation opinions as to the twelve plaintiffs, citing his lack of sufficient records to draw reliable conclusions as to specific causation. The Court sees no logic in forcing a proposed expert to testify to conclusions he has publicly rejected and, therefore, accepts Dr. Gad's withdrawal of his specific causation opinions.

Dr. Gad's general causation opinions contained in his 2005 report and offered at the Daubert hearing, though disputed by defendant's experts, track the existing literature on the subject of arsine toxicity. Certainly, hemolysis is prominently featured in Caravati's discussion of arsine toxicity. See Caravati, supra, at 1393 ("Arsine . . . is the most potent hemolytic agent encountered in industry . . . ."); id. at 1400 ("In arsine gas exposure, the primary target organ is the red blood cell."); id. ("For arsine gas, the major toxicity and cause of mortality is red cell hemolysis.").[20] Caravati does recognize, however, the possibility of direct organ injury from arsine gas. See id. at 1396 ("Arsine may also have a direct toxic effect on the kidney."), as do the MMGs issued by the ATSDR.[21] Moreover, Dr. Gad accurately reports that Caravati identifies a potential for delayed hemolysis. Id. at 1393. Defendant's experts reject both of these claims, but the resolution of such a dispute is not part of the gatekeeping function performed by this Court pursuant to Daubert. Daubert, 509 U.S. at 596 ("Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence.").

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[20] Even Dr. Gad acknowledged the centrality of hemolysis in arsine toxicity. Defendant's Primer, Ex. 2, Gad Deposition, at 126-27 ("What will happen is initially after an exposure, the first place you're going to tend to find the arsine and arsine effect is going to tend to be the red blood cells, and over time it's going to distribute itself to other tissues and eventually be eliminated from the body.").

[21] Having reviewed the literature cited by plaintiffs' experts, the Court suspects that those experts may well be misinterpreting the meaning of "direct injury" to the kidney. As the Court reads the existing literature, direct injury refers to injury that occurs not in the absence of hemolysis, but in addition to the kidney injury caused by the filtering through the kidney of the byproducts of lysed red blood cells. That noted, the Court nevertheless recognizes that such a determination is well outside the scope of its expertise and that its concern is with the experts' methodology, not the substance of their opinions.

Accordingly, the Court determines that Dr. Gad reached his general causation opinions on the toxicity of arsine in a reliable manner, formulating his views on the basis of existing literature on arsine toxicity. Dr. Gad may testify to the general causation opinions contained in his 2005 report, save those opinions relating to chronic exposure to arsine gas.

IV.

IT IS THEREFORE ORDERED that defendant's motion to strike the testimony of Dr. Shayne Gad (Dkt. # 52) is hereby **granted in part** and **denied in part** as outlined above. Defendant's appeal of magistrate judge's order on defendant's motion to strike (Dkt. # 85) is **denied**. Defendant's motion to strike the testimony of Dr. Richard Hastings for failing to file a complete case list (Dkt. # 56) is **denied as moot**.

IT IS FURTHER ORDERED that defendant's request (included in (Dkt. ## 88, 89-104, 110) to exclude the testimony of all three plaintiffs' experts pursuant to Rule 702 of the Federal Rules of Evidence is hereby **granted in part** and **denied in part**. Plaintiffs' experts' testimony will be limited as follows: Dr. Richard Hastings may offer testimony on the objective findings of his physical examinations of plaintiffs. He is not permitted to testify, however, as to either general or specific causation. Dr. Robert Harrison may not testify as to any of the causation opinions developed in this case. Dr. Shayne Gad may testify to the general causation opinions contained in his 2005 report, but may not opine on any chronic exposure theory offered in that report.

IT IS SO ORDERED this 28th day of December, 2005.

Claire V Eagan

CLAIRE V. EAGAN, CHIEF JUDGE
UNITED STATES DISTRICT COURT